

Endovascular Exclusion of the Anterior Communicating Artery with Flow-Diverter Stents as an Emergency Treatment for Blister-Like Intracranial Aneurysms

A Case Report

AYMERIC ROUCHAUD, SUZANA SALEME, BENJAMIN GORY, DAVID AYOUB, CHARBEL MOUNAYER

Department of Interventional Neuroradiology, CHU Dupuytren, Limoges, France.

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Summary

Blood blister-like aneurysms (BLAs) are rare lesions, associated with diffuse subarachnoid hemorrhage (SAH). BLAs tend to rebleed quickly after first bleeding and must be treated as an emergency. Acute treatment is challenging using surgical and endovascular approaches due to the fragile aneurysm wall and small sac. Flow-diverter stents (FDSs) may offer a new option for the treatment of difficult small aneurysms. We describe a case of a ruptured BLA on the anterior communicating artery (ACoMA) treated in the acute phase of SAH by endovascular exclusion of the ACoMA with deployment of two FDSs in the A1/A2 junctions of both anterior cerebral arteries (ACAs). A 61-year-old man was admitted for diffuse SAH with a focal interhemispheric hematoma. Angiography revealed multiple arterial wall irregularities on the ACoMA and both ACAs. We performed an endovascular shunt of the ACoMA by deploying two FDSs in both A1/A2 junctions. Immediate control injections confirmed flow diversion in the A1/A2 segments of the ACAs with decreased blood flow in the ACoMA. The patient's course in hospital was uneventful. A three-month follow-up angiogram confirmed complete exclusion of the aneurysms, complete exclusion of the

ACoMA, and patency of the two ACAs without any persistent arterial wall irregularity. Endovascular bypass using an FDS for a ruptured BLA has never been described. It establishes a new therapeutic option despite the need for antiplatelet therapy. Endovascular ACoMA exclusion using an FDS may be a solution when no other treatment is available for a ruptured BLA.

Introduction

Blood blister-like aneurysms (BLAs) are rare lesions, which can sometimes be difficult to recognize and in most cases are associated with diffuse subarachnoid hemorrhage (SAH)^{1,2}. These aneurysms tend to rebleed quickly after first bleeding and must be treated as an emergency³.

Acute treatment of a ruptured intracranial BLA is challenging using surgical and endovascular approaches²⁻⁵. When occlusion of the parent vessel is not possible, choosing the right endovascular treatment is difficult because there is no consensus on the best management strategy. Coiling of BLA is a high-risk procedure⁶ as the fragile aneurysm wall can easily rupture during catheterization or coil insertion¹. Furthermore, the small sac and wide-neck

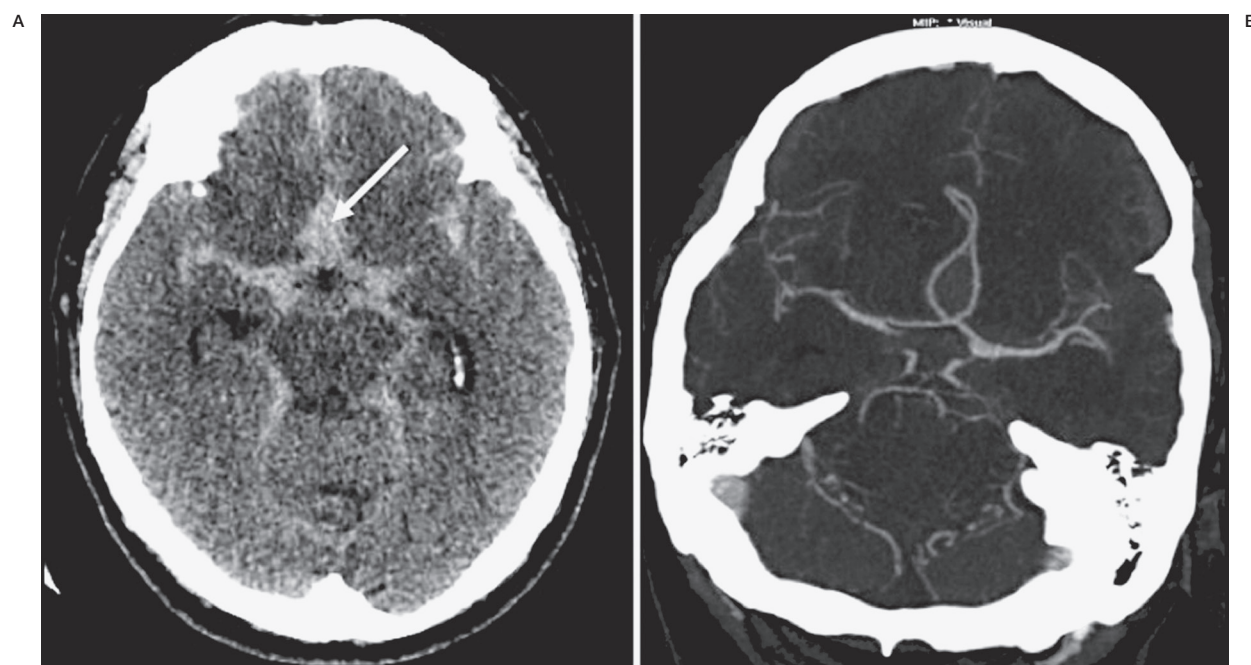


Figure 1 A) Initial non-contrast computed tomography (CT) scan on presentation, showing diffuse subarachnoid hemorrhage with anterior interhemispheric hematoma (arrow). B) CT angiography axial reformat image showing no aneurysm.

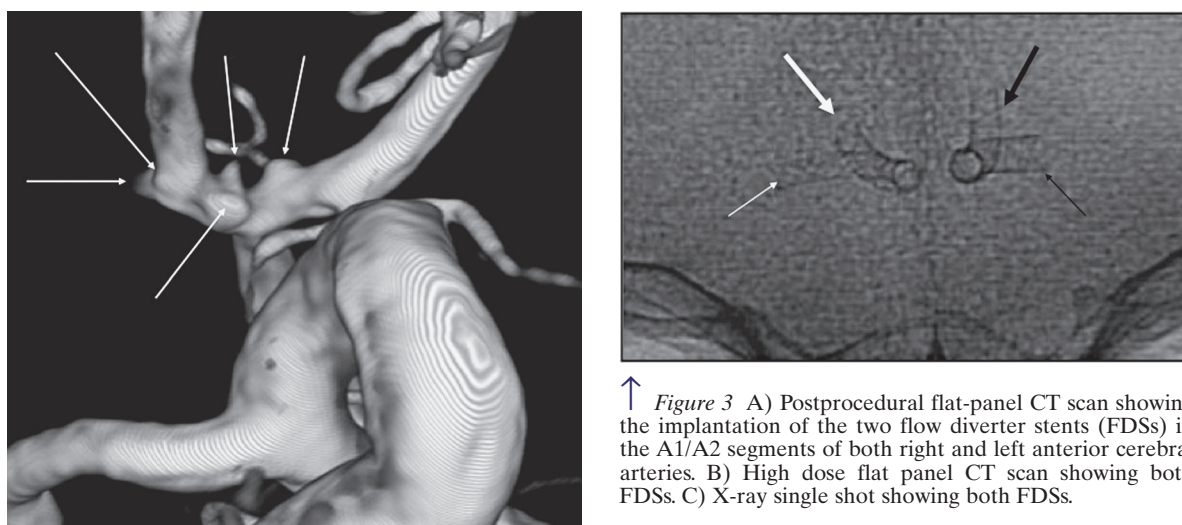


Figure 2 Three-dimensional digital subtraction angiography of the left internal carotid artery showing multiple irregularities (arrows) on the anterior communicating artery and on the A1/A2 junctions on both right and left sides.

Figure 3 A) Postprocedural flat-panel CT scan showing the implantation of the two flow diverter stents (FDSs) in the A1/A2 segments of both right and left anterior cerebral arteries. B) High dose flat panel CT scan showing both FDSs. C) X-ray single shot showing both FDSs.

anatomy make coil placement difficult, enhancing the risk of thromboembolic complications¹.

Improvements in endovascular techniques, with the development of flow-diverter stents (FDSs), could offer a new option for the treatment of difficult small aneurysms, even if the safety and efficiency of these stents are based only on small retrospective series^{7,8}. Princiotta et al. recently described two patients treated

for blood BLAs in two stages, with stent-assisted coiling followed by FDS in-stent insertion for reconstruction of the parent artery⁸. When initial coiling is not possible due to unfavorable anatomy, an FDS can be the first option for reconstructing the parent vessel⁹. These options with stent placement in the acute phase of SAH involve dealing with antiplatelet and anticoagulant therapies and balancing hemorrhagic and

thromboembolic risks¹⁰. If a reconstruction strategy is not suitable due to multiple aneurysms or insertion on a bifurcation, a deconstruction option must be considered. The aim of this paper is to describe a case of a ruptured BLA on the anterior communicating artery (ACoM) treated in the acute phase of SAH by endovascular exclusion of the ACoM with deployment of two FDSs in the A1/A2 junctions of both anterior cerebral arteries (ACAs).

Case Report

A 61-year-old man with no medical history or vascular risk factors was admitted to our institution for headache and vomiting. His Glasgow Coma Scale score was 15. A computed tomography (CT) scan showed diffuse SAH (Fisher grade III, World Federation of Neurosurgeons grade I) with a focal interhemispheric hematoma, without ventricular dilatation (Figure 1A). CT angiography did not highlight any saccular aneurysm (Figure 1B). Three-dimensional rotational angiography revealed multiple arterial wall irregularities located on the ACoM and on the right and left A1/A2 junctions of the ACAs (Figure 2). All arterial wall irregularities measured < 1 mm, with no well-defined neck. These irregularities were considered to be BLAs because they were small in size and because intracranial dissection (another possible diagnosis) does not involve different arterial segments at the same time. One of these irregularities had to be considered as the ruptured BLA, but there was no means of identifying which one corresponded to the bleeding lesion.

Selective neurosurgical or endovascular exclusion of each aneurysm was not possible because they were too small and had unfavorable anatomy. Occlusion of the ACoM with clipping or coiling was not considered a good option because the target BLAs were located not only on the ACoM but also on the right and left A1/A2 segments, and such a procedure could not protect irregularities located on the ACAs. We were facing a challenge: we had to exclude all these arterial irregularities without shunting the ACA territories.

We decided to perform endovascular exclusion of the ACoM by the deployment of two 2.5 × 20 mm FDSs (Pipeline Embolization Device; ev3 Endovascular Inc., Plymouth, MN, USA) in both right and left A1/A2 junctions. This option could not only exclude the ACoM but also reconstruct the arterial wall of

both ACAs and protect all the BLAs (Figure 3). The procedure was performed under general anesthesia with systemic heparinization (8000 IU during the procedure). The patient was loaded with 250 mg aspirin just before the first stent deployment and abciximab (7.2 mg/h) was started just after the deployment of the first stent and continued for 12 hours, followed by dual antiplatelet therapy for three months and aspirin for one year after the procedure. Antiplatelet therapy is challenging in case of acute intracranial hemorrhage because of the possible need for external ventricular derivation (EVD), but in this case there was no ventricular dilatation, so EVD was not required and there was no contraindication for antiplatelet treatment.

Immediate control injections confirmed flow diversion in the A1/A2 segments of the ACAs with decreased blood flow in the ACoM. Flow competition was confirmed by the washing flow coming from the contralateral side (Figure 4). The patient's course in hospital was uneventful with no rebleeding, vasospasm, or ischemic lesions in the shunted territories. A three-month follow-up catheter angiogram confirmed complete exclusion of the aneurysms, complete exclusion of the ACoM, and patency of the two ACAs (Figures 5 and 6) without any persistent arterial wall irregularity.

Discussion

Acutely ruptured intracranial BLAs are unstable and have a high tendency to rebleed in the acute phase³ – more so than other types of aneurysms. Protecting the patient against further bleeding has to be the goal of treatment. Due to their small size and fragile wall, selective treatment of these lesions is very difficult to perform using endovascular or surgical approaches. Most previous studies have reported BLAs on the non-branching supraclinoid portion of the internal carotid artery – the most common location for these lesions². These rare lesions are challenging in terms of treatment but also diagnosis, as previously reported by Gaughen et al.¹¹. BLA remains an elusive diagnosis, particularly using CT angiography, which does not have 100% sensitivity¹¹. Thus, in the face of SAH and otherwise negative CT angiogram findings, a catheter cerebral angiogram should be obtained to absolutely exclude a BLA.

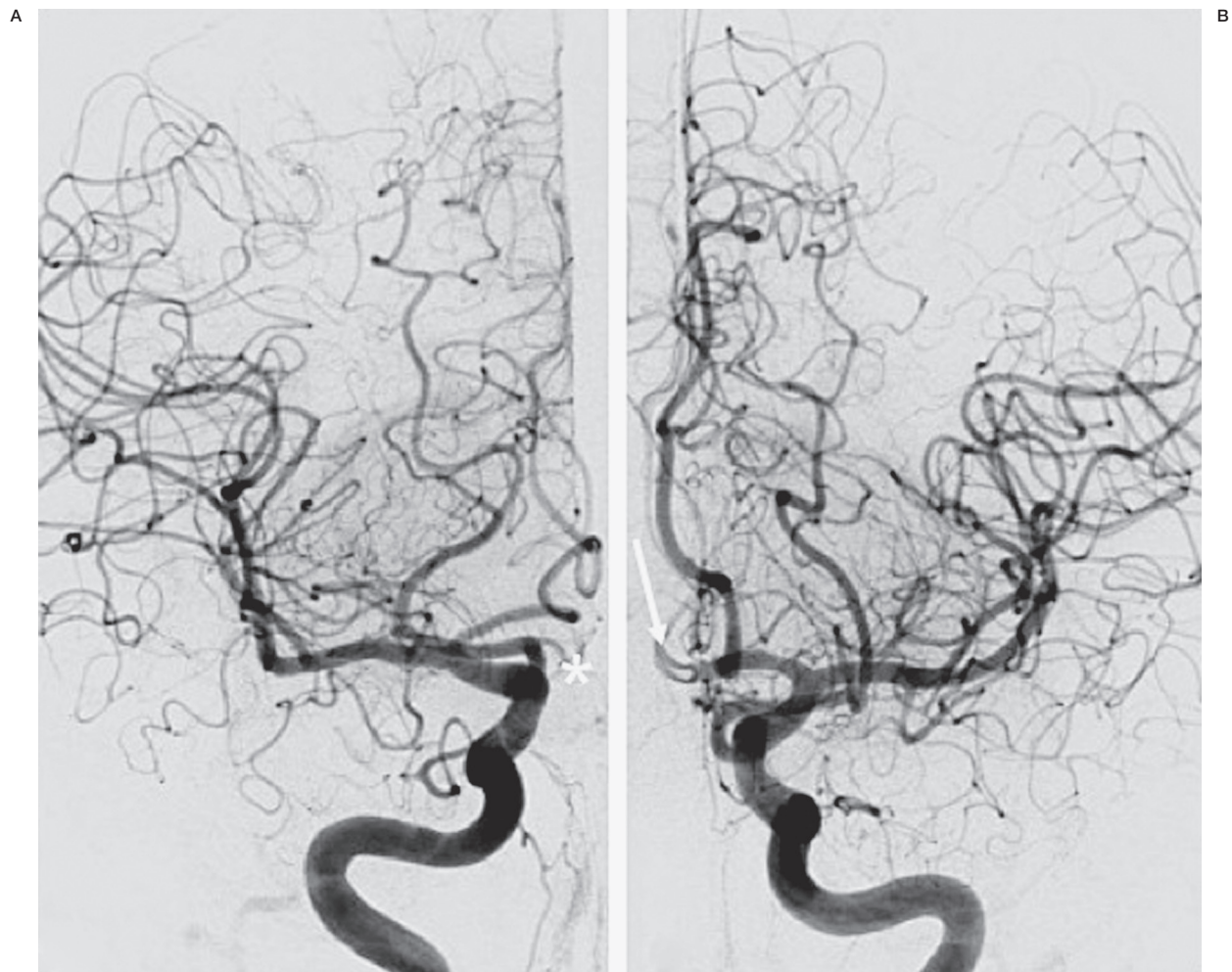


Figure 4 Postoperative digital subtraction angiography in the anteroposterior projection showing the flow competition in the anterior communicating artery (ACoM). A) No filling of the ACoM by the right internal carotid artery (*). B) Persistence of low flow in the ACoM by the left internal carotid artery (arrow).

Regarding treatment, parent vessel occlusion remains the simplest treatment, using either the endovascular or surgical approach when possible¹². Surgical trapping¹³⁻¹⁵ and stent-assisted endovascular coiling¹⁶⁻¹⁹ have also been reported.

In the present case, several sidewall irregularities were detected on the ACoM complex, including the ACoM and both ACAs at the A1/A2 junctions. One of these irregularities had to be considered the culprit ruptured BLA, but it was impossible to determine which lesion had bled (Figure 2). Exclusion of all the lesions from the blood circulation was the only way to guarantee that the ruptured BLA had been excluded.

Surgical treatment was discussed. The advantage of the surgical approach was that it might

have enabled the BLA that had bled to be identified, but it would not have been possible to achieve selective treatment of each aneurysm and clipping of the ACoM would not have been sufficient, as it would not have protected BLAs located on both sides of the A1/A2 segments. In addition, endovascular occlusion of the ACoM with coiling would not have protected irregularities located on the ACAs and selective coiling was impossible to perform.

Recently, FDSs have been employed in the acute phase of SAH to reconstruct the vessel wall and cure the aneurysms, with favorable results reported in small series or case reports^{8,9,20-24}. However, given the location of the multiple lesions in our patient, using a unique FDS in the ACoM would not have covered all the

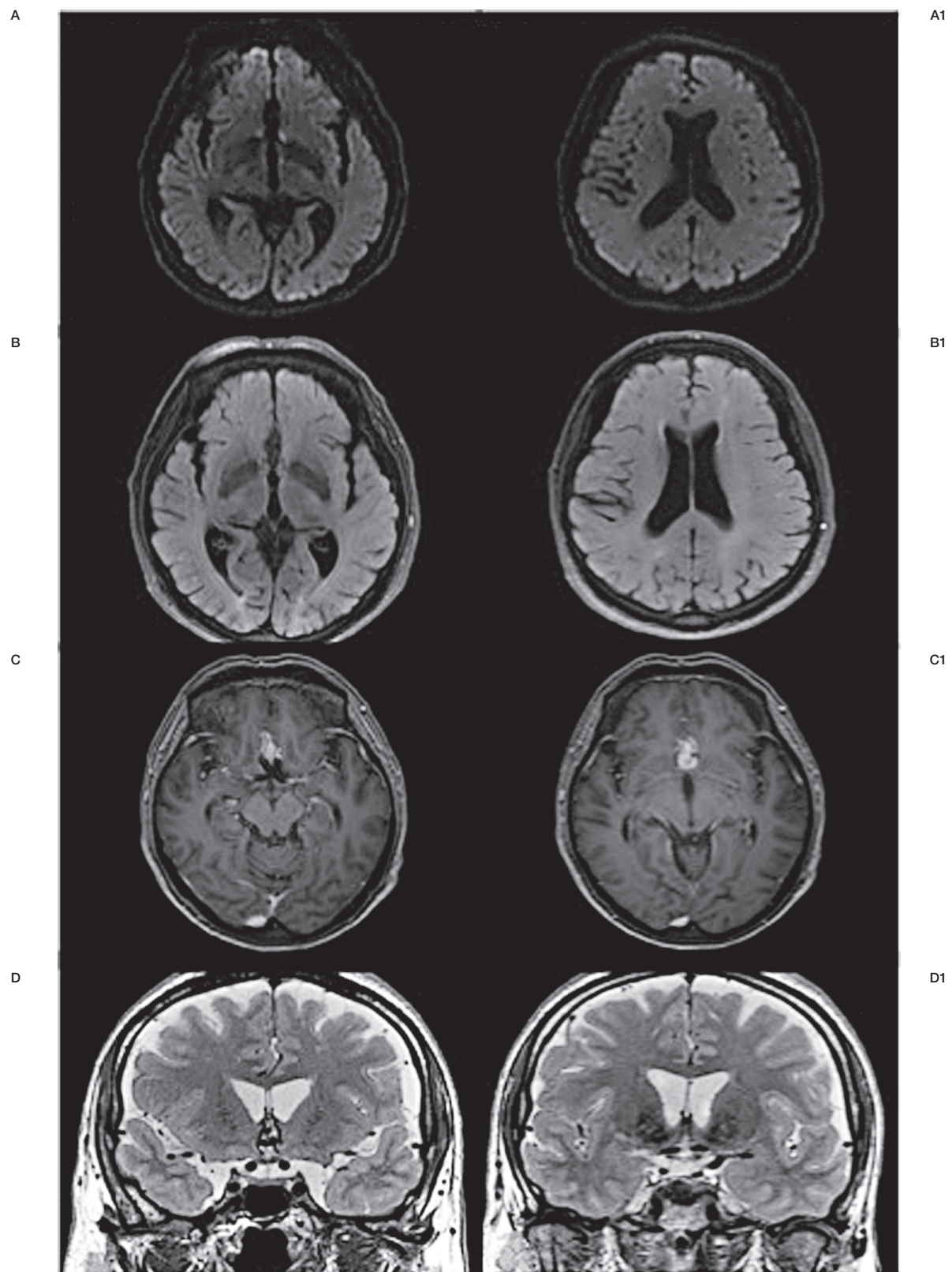
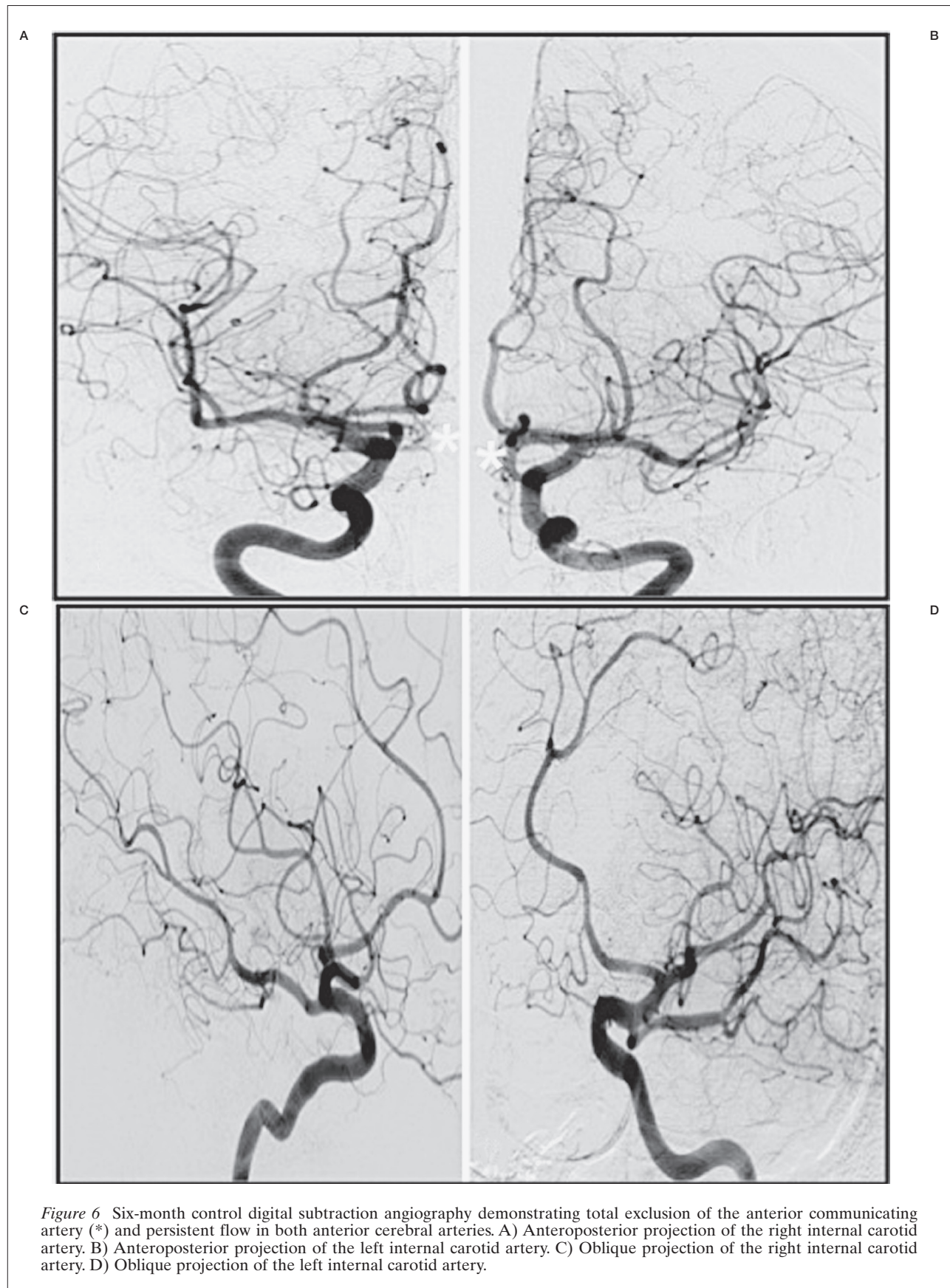


Figure 5 Postoperative magnetic resonance imaging scan showing no ischemic lesion in the shunted territories of the anterior communicating artery. A) DWI. B) FLAIR. C) Gadolinium-enhanced T1. D) T2.



lesions and one of the ACAs would have been shunted. Endovascular treatment of aneurysms located on the AComA using X-configured cross-kissing stents has been reported^{19,25}, but this alternative was not suitable in our conformation as FDS meshes are too dense to allow cross-kissing. Stent implantation involves antiplatelet therapy, which constitutes a challenge in case of acute intracranial hemorrhage. The main risk of complication secondary to antiplatelet therapy in the acute phase of intracranial hemorrhage is due to EVD. If EVD is required for ventricular dilatation, it should be done before antiplatelet therapy is started and hence before stent deployment.

Our therapeutic option used the principle of the FDS, according to Fiorella et al.²⁶. When a vessel shunted by the FDS does not supply a parenchymal territory it will be occluded as well as the covered aneurysm. We decided to perform an endovascular AComA endovascular exclusion using two FDSs positioned in both ACAs at the A1/A2 junction level. As the AComA was totally shunted, all the lesions were either covered or shunted and the flow in both ACAs was preserved, whereas there was a drastic reduction of the flow in the AComA (Figures 3 and 4). The AComA occlusion was an expected phenomenon related to the flow

competition induced in both ACAs by FDS use. In order to ensure that the endovascular exclusion of the AComA was not responsible for ischemic lesions, a postoperative magnetic resonance imaging scan with a diffusion-weighted imaging sequence was performed. Despite occlusion of the AComA, no ischemic lesion in the shunted territories (i.e. Heubner's artery, the medial lenticulostriate artery territory and the subcallosal area) was observed (Figure 5). The three-month follow-up control confirmed our hypothesis, showing the total angiographic disappearance of the AComA and the regular arterial wall of both ACAs (Figure 6).

To the best of our knowledge, endovascular bypass with FDSs for a ruptured BLA has never been described and establishes a new therapeutic option despite the need for anti-platelet therapy, which remains a concern in patients presenting with acute SAH.

Conclusion

Endovascular communicating artery exclusion using FDSs is a new and original therapeutic option, which can be a solution in cases when no other treatment is available for ruptured BLA.

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Charbel Mounayer, MD, PhD
 Department of Interventional Neuroradiology
 CHU Dupuytren, 2 Avenue Martin Luther King
 Limoges, France
 Tel.: +33 5 55 05 80 11
 Fax: +33 5 55 05 62 88
 E-mail: cmounayer@hotmail.com